# Diagnostic value of vectorcardiogram in strictly posterior infarction[1]

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*In 313 patients undergoing selective coronary angiography for chest pain, the electrocardiogram and vectorcardiogram were studied for evidence of posterior myocardial infarction. Electrocardiographic evidence for posterior infarction was an $R/S$ ratio equal to or greater than 1 in $V_4R$ and $V_1$. Vectorcardiographic criteria for posterior wall infarction were defined in the horizontal plane by either counterclockwise rotation with an anteriorly oriented 0·04 second vector and mean QRS axis or clockwise rotation in the absence of right ventricular hypertrophy. Right ventricular hypertrophy was excluded by clinical evaluation, chest x-ray, and cardiac catheterization. Moderate to severe obstruction of the right coronary artery and/or left circumflex coronary artery or posterior wall dyssynergy was considered supportive angiographic evidence of a posterior myocardial infarction. Of 31 patients with vectorcardiograms diagnostic of strictly posterior myocardial infarction, 25 had associated coronary disease. Only 6 of the 31 patients had electrocardiographic evidence of strictly posterior myocardial infarction and only 4 of the 6 patients had abnormal coronary angiograms. The vectorcardiogram appears to be more reliable than the electrocardiogram in the diagnosis of strictly posterior infarction.*

The diagnosis of myocardial infarction involving the true posterior wall has always been difficult. The primary electrocardiographic criterion of posterior infarction is a prominent R wave in the anterior praecordial leads, but it is frequently absent or equivocal (Toutouzas *et al.*, 1969). It is not uncommon for a strictly posterior myocardial infarction to coexist with or masquerade as a complete right bundle-branch block (Doucet, Walsh, and Massie, 1965; Lipman and Massie, 1965). Though the advantage of a vectorcardiogram in clinical cardiology has been questioned, it has been suggested that the vectorcardiogram may offer a distinct advantage in the diagnosis of strictly posterior infarction (Massie and Walsh, 1960).

Since isolated posterior infarctions are not common and rarely cause death, necropsy verification of isolated posterior infarction is difficult to obtain. Similarly clinical correlation with acute myocardial infarction is difficult, since such a diagnosis depends on electrocardiographic change. Selective coronary angiography provides an independent, readily

available, and accurate means of correlating anatomical change with the electrocardiogram and vectorcardiogram (Lee *et al.*, 1968). Though the mere presence of coronary atherosclerosis does not necessarily imply that myocardial infarction has occurred, it does provide a reasonable basis for exclusion of false positive electrocardiograms and vectorcardiograms.

In this study, the vectorcardiogram and electrocardiogram were correlated with the coronary arteriogram and left ventriculogram to determine if the vectorcardiogram was more reliable than the electrocardiogram in the diagnosis of strictly posterior myocardial infarctions.

## Subjects and methods

Studies were made of 313 consecutive unselected patients undergoing selective coronary angiography for the evaluation of chest pain. Patients with mitral stenosis, primary pulmonary hypertension, or congenital heart disease were not included in this study. Right ventricular hypertrophy was excluded by clinical evaluation, chest x-ray, and right heart catheterization. All patients received a vectorcardiogram and electrocardiogram one day before the coronary arteriogram. Vectorcardiograms were recorded on a Hewlett-

TABLE  *Clinical summary of 25 patients with coronary disease*

| Case No. | Age (yr) | Sex | Coronary angiogram | | | Left ventriculogram posterior wall dyssynergy | Electrocardiogram QRS morphology | Vectorcardiogram QRS loop |
|---|---|---|---|---|---|---|---|---|
| | | | Right coronary | Left circumflex | Left anterior descending | | | |
| 1 | 40 | M | 3+ | 3+ | 3+ | Present | Normal | Post. wall infarct. |
| 2 | 42 | M | 3+ | 3+ | 3+ | Present | Normal | Post. wall infarct. |
| 3 | 62 | M | 3+ | 3+ | 3+ | Present | Normal | Post. wall infarct. |
| 4 | 46 | F | 2+ | 2+ | 2+ | Present | Normal | Post. wall infarct. |
| 5 | 57 | M | 3+ | 3+ | 3+ | Present | Normal | Post. wall infarct. |
| 6 | 65 | M | 3+ | 3+ | 3+ | Absent | Normal | Post. wall infarct. |
| 7 | 60 | M | 3+ | 3+ | 3+ | Absent | Normal | Post. wall infarct. |
| 8 | 61 | M | 3+ | 3+ | 3+ | Absent | Normal | Post. wall infarct. |
| 9 | 64 | M | 3+* | 3+ | 3+ | Not done | Normal | Post. wall infarct. |
| 10 | 50 | M | 3+ | 3+ | 3+ | Not done | Normal | Post. wall infarct. |
| 11 | 59 | M | 3+ | 2+ | 3+ | Not done | Normal | Post. wall infarct. |
| 12 | 50 | M | 3+ | 1+ | 1+ | Absent | RBBB | Post. wall infarct. + RBBB |
| 13 | 48 | M | 3+ | 3+ | 3+ | Present | Normal | Post. and infer. wall infarcts |
| 14 | 43 | F | 3+ | 1+ | 1+ | Absent | Normal | Post. and infer. wall infarcts |
| 15 | 54 | M | 3+ | 3+ | 3+ | Not done | Normal | Post. and infer. wall infarcts |
| 16 | 61 | M | 3+ | 3+ | 3+ | Not done | Possible infer. wall infarct. | Post. and infer. wall infarcts |
| 17 | 67 | M | 3+ | 3+ | 3+ | Present | Infer. wall infarct. | Post. and infer. wall infarcts |
| 18 | 47 | M | 3+ | 1+ | 1+ | Not done | Infer. wall infarct. | Post. and infer. wall infarcts |
| 19 | 42 | M | 0 | 3+ | 1+ | Present | Infer. wall infarct. | Post. and infer. wall infarcts |
| 20 | 50 | M | 3+ | 2+ | 2+ | Not done | Infer. wall infarct. | Post. and infer. wall infarcts |
| 21 | 52 | M | 2+ | 2+ | 2+ | Absent | Post. wall infarct. | Post. infer., and lateral wall infarcts |
| 22 | 48 | M | 3+ | 3+ | 3+ | Present | Post., infer., and lateral wall infarcts | Post. infer., and lateral wall infarcts |
| 23 | 62 | M | 3+ | 3+ | 3+ | Present | RBBB, anter., infer., and lateral wall infarcts | Post., anter., infer., and lateral wall infarcts Conduction delay |
| 24 | 55 | M | 3+ | 3+ | 1+ | Present | Post. wall infarct. | Post. and lateral wall infarcts |
| 25 | 45 | M | 3+ | 3+ | 3+ | Absent | Post. wall infarct. | Post. and lateral wall infarcts |

* Necropsy.
RBBB Complete right bundle-branch block.



FIG. I  *Relation of vectorcardiogram and electrocardiogram in typical strictly posterior infarction showing abnormal vectorcardiogram and normal chest leads of electrocardiogram.*
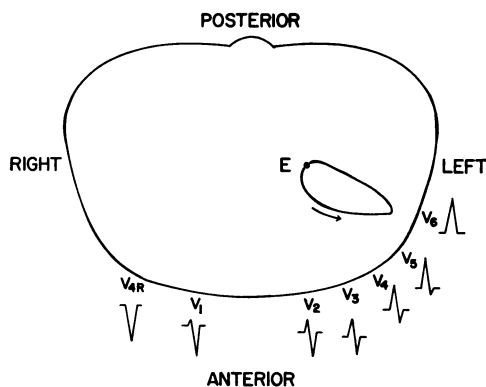
Packard 157A Vector Programmer using the Frank (1956) lead system. The vectorcardiograms were obtained in the horizontal, left sagittal, and frontal planes at 0·5 and 0·1 mV standardizations. Orthogonal leads X, Y, and Z were also obtained. Electrocardiographic criteria for a strictly posterior myocardial infarction were an R/S ratio greater than 1 in V4R and/or V1 or an R/S ratio equal to 1 in both V4R and V1 (Lipman and Massie, 1965). Vectorcardiographic criteria diagnostic of a strictly posterior myocardial infarction were based on prominent anterior forces in the horizontal plane. These were defined by an anteriorly oriented 0·04 sec vector and an anteriorly oriented mean axis in the horizontal plane in the presence of counterclockwise rotation. The presence of clockwise rotation in the horizontal plane was also considered compatible with a posterior infarction in the absence of right ventricular hypertrophy (Chou and Helm, 1967; Hugenholtz, Forkner, and Levine, 1961; Mathur and Levine, 1970). In the orthogonal leads these findings were confirmed by predominantly anterior or positive QRS forces in the Z lead. The vectorcardiographic T loops were analysed for evidence of ischaemia using a length-width ratio of less than or equal to 2·6:1 (Gray and Bell, 1971).

Selective coronary arteriograms were performed using the Judkins' technique (1967). Cine films

were recorded in several planes on 16 mm or 35 mm film. The coronary arteriograms were classified according to Demany, Tambe, and Zimmerman (1967), with 0 equalling normal; 1+, mild or less than 50 per cent narrowing of a coronary artery; 2+, moderate or 50 per cent obstruction of the vessel lumen; and 3+, severe disease or greater than 50 per cent obstruction of each major coronary artery. Patients with less than 2+ disease or equivocal angiograms were excluded. Left ventricular cineangiography was performed in the right anterior oblique position. A 50 ml bolus of sodium and meglumine diatrizoate was injected through an 8 French pigtail catheter situated in the left ventricle. Left ventricular angiography was not done in some patients because of technical difficulties.

The left ventriculogram was evaluated for evidence of posterior wall dyssynergy (Herman and Gorlin, 1969) which was defined as a consistent abnormality in the equality and force of contraction of the posterior wall in the absence of premature ventricular contractions. A myocardial infarction of the true posterior wall was assumed to be present if there was posterior wall dyssynergy or moderate to severe coronary disease of the posterior vessels. Since both the right and left circumflex coronary artery supply the posterior wall, a complete or almost complete occlusion (3+) of the right and/or left circumflex coronary artery or moderate coronary artery disease (2+) of both the right and left circumflex coronary arteries satisfied the criteria of significant coronary artery disease of the posterior wall (Proudfit, Shirey, and Sones, 1966).

## Results

There were 31 patients whose vectorcardiograms were diagnostic of a strictly posterior infarction. Of these there were 25 who demonstrated coronary artery disease of the posterior wall on the angiograms. There were 6 of 31 patients whose electrocardiograms were diagnostic of a posterior myocardial infarction, and, of these, 4 had associated angiographic evidence of coronary artery disease of the posterior wall. There were 2 patients with severe coronary disease who had a complete right bundle-branch block on the electrocardiogram in which the vectorcardiogram demonstrated additional diagnostic evidence of posterior myocardial infarction.

Fifteen patients with vectorcardiographic evidence of a strictly posterior myocardial infarction and angiographic evidence of coronary disease had no diagnostic evidence of infarction on the electrocardiogram. Of these, 14 had completely normal QRS complexes and one patient had a complete right bundle-branch block. The data on the 25 patients with coronary artery disease are summarized in the Table.

There were 4 false positive vectorcardiograms in patients with normal coronary angiograms in which the electrocardiogram correctly did not show a posterior myocardial infarction. Two of these electrocardiograms had other diagnostic evidence of ischaemic heart disease, one with an acute subendocardial infarction and one with an old inferior infarction. The other two electrocardiograms were normal.

The 0·04 sec vector of the 31 patients studied ranged from 5 degrees to 205 degrees, with a mean of 28 degrees. The mean (half area) horizontal plane QRS vector ranged from 0 degrees to 190 degrees with a mean of 27 degrees. One patient did not have an anterior mean QRS axis in the horizontal plane but had both vectorcardiographic and electrocardiographic evidence of a posteroinferolateral myocardial infarction and severe three-vessel coronary artery disease on the angiograms.

## Discussion

Theoretically the vectorcardiogram should be superior to the electrocardiogram in the diagnosis of strictly posterior myocardial infarction, since such infarctions usually shift the horizontal plane vector anteriorly preserving the normal counterclockwise rotation (Hugenholtz *et al.*, 1961). This occurred in 21 of 25 patients with coronary disease in this study. Fig. 1 is a diagram of the usual relation of the electrocardiogram to the vectorcardiogram in the presence of a strictly posterior infarction. The shift in the electrical forces anterior to the E point is not always recorded in the conventional chest leads on the electrocardiogram. The typical vectorcardiographic appearance of a strictly posterior infarction (an anterior 0·04 sec vector, mean horizontal QRS axis in the left anterior quadrant, and either counterclockwise rotation or figure-of-eight rotation in the horizontal plane) was not associated with diagnostic electrocardiographic changes in any patient in this study (Fig. 2 and 3). On the basis of this study the electrocardiographic appearance of a posterior myocardial infarction seems unlikely unless there is associated inferior and/or lateral wall infarction (Toutouzas *et al.*, 1969).

When there is counterclockwise rotation in the horizontal plane, it is unlikely that the scalar electrocardiogram will demonstrate a prominent R wave in the anterior praecordial leads unless the initial forces are oriented anteriorly and to the right as occurs in lateral wall infarction. Two of the electrocardiograms compatible with posterior infarction in pa-

tients with coronary disease had vectorcardio-grams with counterclockwise rotation in the horizontal plane and a prominent initial force in the right anterior quadrant. The 0·02 sec vector was to the right of 110 degrees and the 0·025 to 0·03 sec vector in the frontal plane was superiorly oriented compatible with a postero-infero-lateral infarction (Fig. 4). There were 4 patients with coronary disease, whose electrocardiograms were abnormal, with clockwise rotation in the horizontal plane. Two of these patients had a prominent R wave in V4R and V2, with vector loops demon-strating terminal forces in the right posterior quadrant compatible with posterolateral in-farction (Fig. 5). The other 2 patients had a right bundle-branch block on the electro-cardiogram but in addition the vectorcardio-graphic horizontal plane loops were oriented entirely anteriorly consistent with a posterior infarction (Fig. 6).

The aetiology of the false positive electro-cardiograms and vectorcardiograms cannot be explained. Patients with thin chest walls might
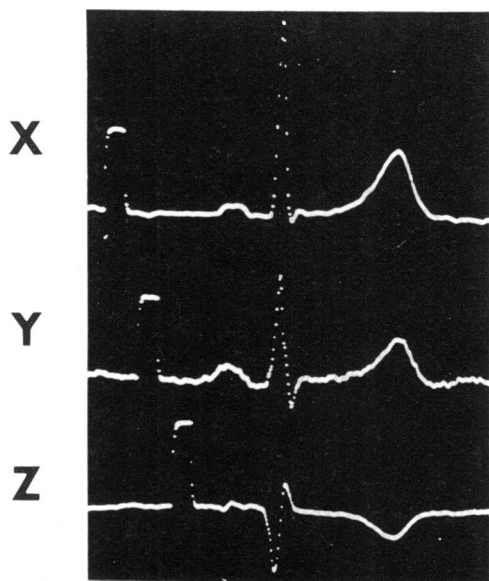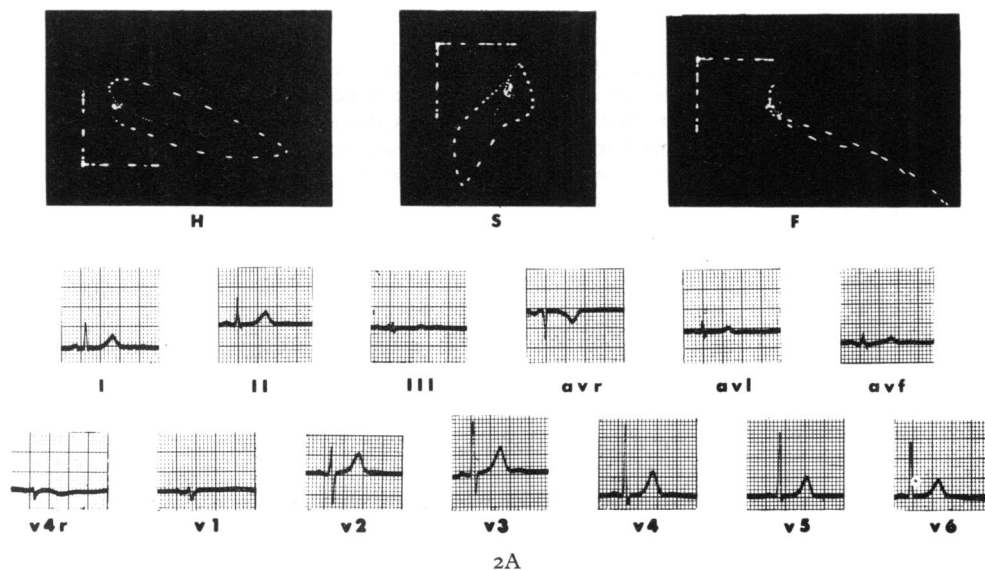


2A



2B

FIG. 2 *(A) Typical vectorcardiogram of strictly posterior infarction and normal electro-cardiogram in a patient with severe coronary disease. (B) Orthogonal leads showing pre-dominantly anterior z lead.*
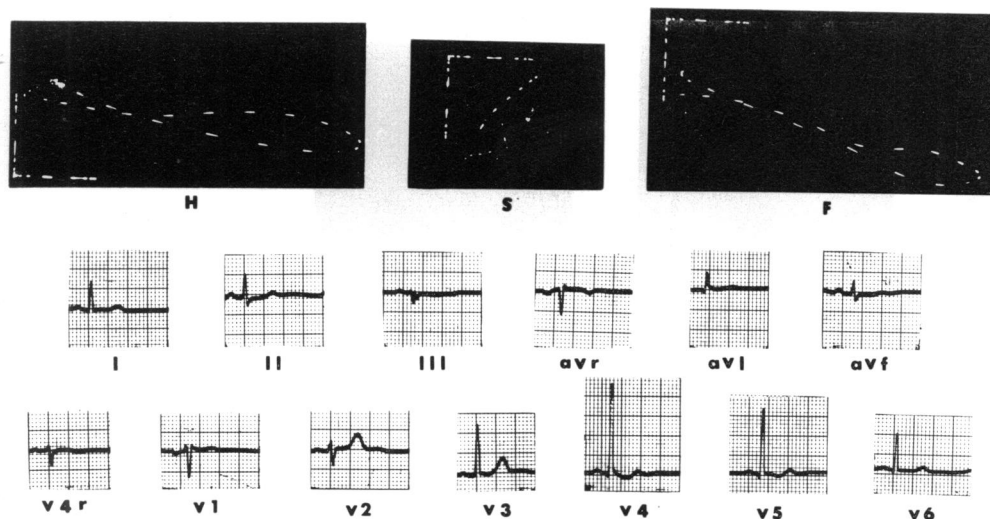
FIG. 3 *Vectorcardiogram diagnostic of posterior infarction with figure-of-eight rotation and normal electrocardiogram in a patient with severe coronary disease.*

theoretically demonstrate a prominent anterior force in the horizontal plane in the absence of posterior wall infarction. None of the patients in this study with false positive vectors had an excessively thin body habitus. One of the false positive electrocardiograms and vectorcardiograms, however, was in a patient with a mild pectus excavatum. It is possible to have a myocardial infarction in the absence of demonstrable coronary disease on the angiogram (Dear *et al.*, 1971). Though this is not likely, it is conceivable that some of the patients might fall into this category. Even though false positives occurred in this study, the correlation of the vectorcardiogram with the angiograms was acceptable. This is especially impressive since there was a lower percentage of false positive vectorcardiograms than electrocardiograms even though the vectorcardiogram was more sensitive. Vectorcardio-

FIG. 4 *Vectorcardiogram with posteroinferolateral infarction and electrocardiogram compatible with posterior infarction in a patient with coronary disease.*
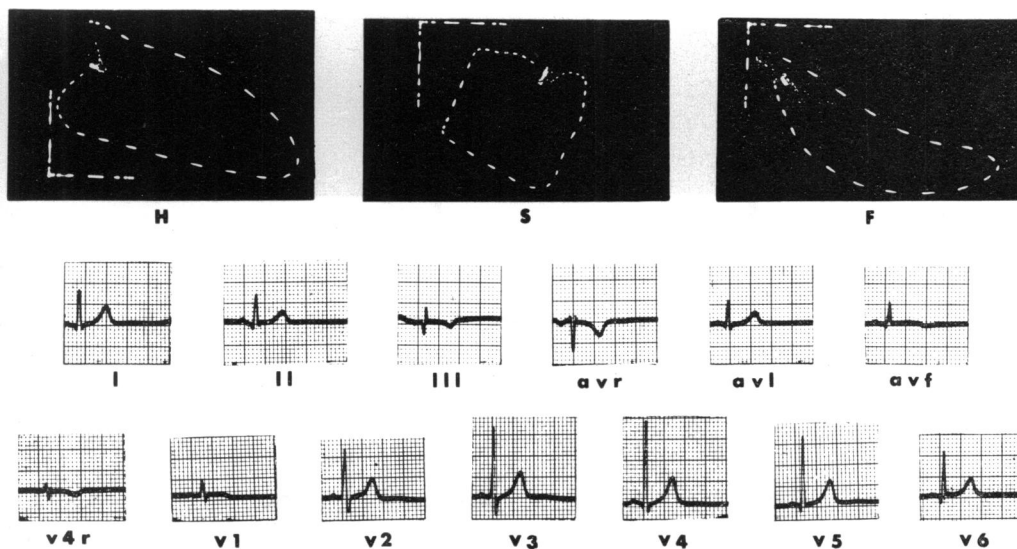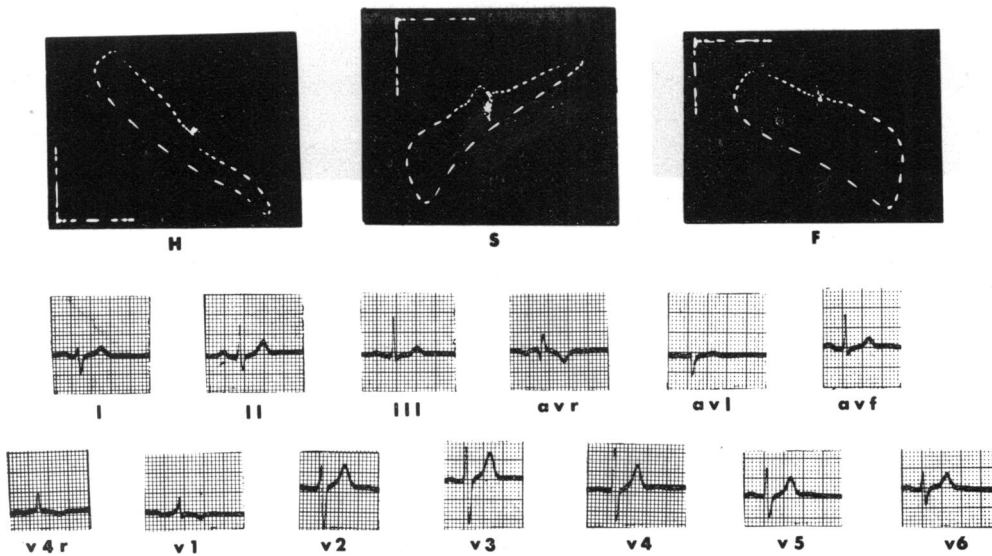
FIG. 5 *Vectorcardiogram showing posterolateral infarction with terminal forces in the right posterior quadrant, with clockwise rotation, and electrocardiogram showing posterior infarction in a patient with severe coronary disease and normal right heart catheterization.*

graphic T loop analysis was not helpful in eliminating false positives (Gray and Bell, 1971).

The infrequency of posterior wall dyssynergy is not unexpected. The true posterior wall is not only technically difficult to visualize in the standard left ventriculogram, but isolated posteroseptal infarction might not pro-

duce a significant degree of dyssynergy. Of 18 patients with coronary disease and technically satisfactory left ventriculograms, 11 demonstrated posterior wall dyssynergy; 6 of these had vectorcardiographic evidence of associated anterior, inferior, or lateral wall infarction.

The most important point is that 14 of the

FIG. 6 *Vectorcardiogram with evidence of both posterior infarction and right bundle-branch block in a patient with severe coronary disease and only right bundle-branch block on the electrocardiogram.*
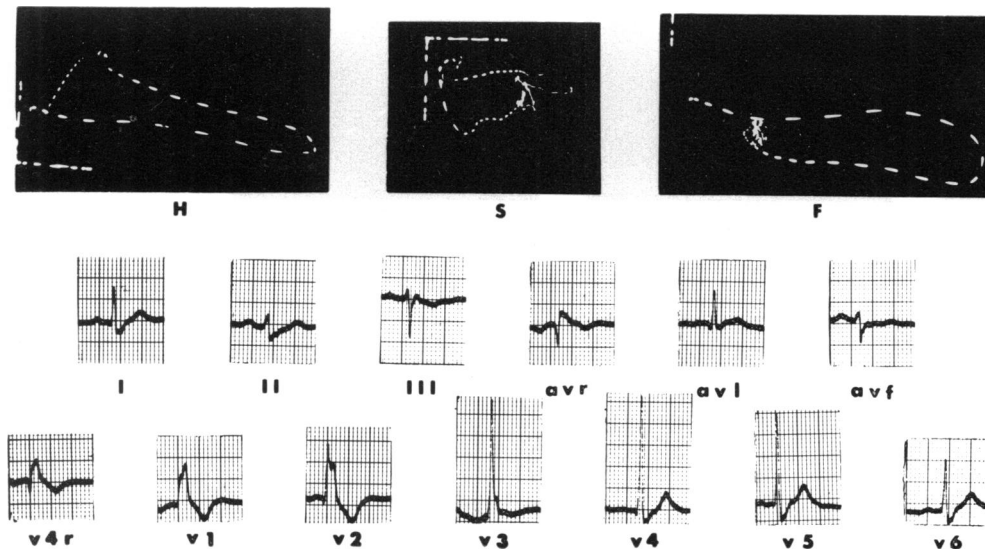
electrocardiograms were normal or nearly normal, but the vectorcardiogram in each case demonstrated a strictly posterior myocardial infarction. There was one patient with vectorcardiographic evidence of a posterior infarction and complete right bundle-branch block but only a right bundle-branch block on the electrocardiogram. The vectorcardiogram was not only correct in determining the presence of a posterior infarction but was the only clinical criterion for the diagnosis of ischaemic heart disease in 15 of the 25 patients with coronary disease included in this study.

The vectorcardiogram is a reliable and accurate method of detecting strictly posterior myocardial infarctions that are frequently not present on the electrocardiogram. The vectorcardiogram is also of value in correctly establishing a diagnosis of ischaemic heart disease in some cases in which the electrocardiogram is normal or not diagnostic. The use of the vectorcardiogram in the evaluation of patients with suspected coronary disease, therefore, seems to be of value. The routine use of three-channel electrocardiographic recording machines equipped with the Frank (1956) lead system for the recording of orthogonal leads seems justifiable. Application of the knowledge that a predominantly anterior force in the Z lead may represent a strictly posterior infarction even in the presence of a normal twelve-lead electrocardiogram appears to be important in establishing future computer programs for the interpretation of electrocardiograms.

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